Hemiballism: report of 25 cases

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Abstract

Twenty three patients with hemiballism and two with biballism were studied. Ischaemic and haemorrhagic strokes were the cause in most patients. Other causes were encephalitis, Sydenham's chorea, systemic lupus erythematosus, basal ganglia calcifications, non-ketotic hyperglycaemia, and tuberous sclerosis. Neuroimaging studies showed a lesion of the subthalamic nucleus in only six patients. In others, different subcortical structures were involved or the results were normal. Only two patients had "pure" hemiballism. The others had other types of dyskinesias, mainly chorea, which was present in 16 patients. The prognosis was usually good.

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Hemiballism is a relatively rare hyperkinetic disorder, characterised by irregular, wide amplitude, vigorous movements of the limbs, primarily due to involuntary activity of the proximal limb and associated axial muscles.[1] Hemiballism was formerly thought to have a poor prognosis with inexorable progression to death within weeks or months, but recent studies have reported a high likelihood of survival and even spontaneous recovery.[2–5] This can be particularly applied to a vascular aetiology of hemiballism.[5]

The most consistent neuropathological finding in hemiballism is a lesion of the contralateral subthalamic nucleus, usually of vascular origin.[6 7] High resolution neuroradiological imaging, however, provided evidence of associated lesions lying elsewhere in the brains of hemiballistic patients (globus pallidus, striatum, thalamus, cerebral cortex, internal capsule, etc).[8–10]

Hemiballism has an incidence in the order of 1 in 500 000 of the general population,[11] which may explain the small series published. In 1947, Whittier[6] found only 60 cases reported worldwide, and Muenter[4] gathered 29 cases seen over a 40 year period at the Mayo Clinic. Dewey and Jankovic[5] identified 21 cases with hemiballism-hemichorea during a nine year period in the Baylor Movement Disorder Clinic, estimating that only 0·7% of patients with movement disorders might have this type of dyskinesia. Herein, we report 25 patients with ballistic movements, focusing on the cause, clinical features and course, neuroradiological findings, and response to treatment.

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Patients and methods

The study comprised 25 patients with ballistic movements diagnosed at the Department of Neurology UCC (Belgrade) from 1983 to 1992. They were all examined by the same neurologist (VK). The onset and mode of disease expression were appraised according to the patient's own statements, or, when this was impossible, from heteroanamnestic data. Other associated types of involuntary movements, neurological deficits, or other relevant clinical findings and information were precisely recorded.

The patients were classified according to the side and affected extremities, presence and nature of other dyskinesias, and to a probable cause of ballism. Degree of disability was rated as mild (rare abnormal movements, not causing functional disability), moderate (occasional functional disability), and severe (considerable functional disability).

Previous medical histories of other disorders and risk factors for cerebrovascular disease (diabetes mellitus, hypertension, hyperlipidaemia, etc) were cautiously revised. CT and MRI studies were obtained whenever possible. Blood counts, thyroid function, serum calcium, potassium, and magnesium, liver and renal function tests, lupus erythematosus test, immunoglobulin concentrations and complement activity in serum, detection of immune complexes and some organ specific autoantibodies were regularly measured. Examination of CSF, including isoelectric focusing with immunofixation for the detection of oligoclonal bands, was conducted in 10 patients. The likely cause of the disorder could be clearly determined in most patients. Stroke was considered as a causal factor despite normal brain imaging studies if the course of the disease and other clinical and laboratory findings indicated so ("assumed stroke").

The patients were regularly followed up at three to six month intervals for between nine months and eight years (mean 2·4 years). The emphasis was on any change in expression of involuntary movements related to any change in their medication.

Results

In 25 patients (seven males and eighteen females) the mean age at onset of ballistic movements was 58·9 (range 15–80) years. The time lapse between the onset of first symptoms and admission to our department varied from a few hours to five months. Two

Table 1 Clinical characteristics of 25 patients with hemiballism

Patient	Age	Sex	Chorea	Other dyskinesias	Involvement	Disability
1	79	F	+	Oromandibular and lingual choreic movements	A, Le, Fo, Fa	Severe
2	66	F	−	Orobuccal twitching	A, Fa	Mild
3	59	M	+	Dystonia	A, Le, H, Fo	Severe
4	15	F	+	Dystonia, athetosis	A, Le, H, Fo, Fa	Moderate
5	68	M	−	None	A, Le	Severe
6	78	F	−	Orobucal movements	A, Le, Fa	Severe
7	56	F	−	Dystonia, oromandibular—lingual movements	A, Le, Fa, Tr	Moderate
8	77	M	+	None	A, Le, H, Fo	Severe
9	66	F	+	Dystonia, athetosis, right facial grimacing	A, H, N, Fa	Mild
10	19	F	−	Orobuccal movements	A, Le, Fa	Severe
11	58	F	+	Dystonia	A, Le, N	Moderate
12	62	F	+	None	A, Le, H, Fo	Severe
13	80	F	+	Orobuccal movements	A, Le, H, Fo, Fa	Severe
14	60	F	+	None	A, Le, H, Fo	Severe
15	35	F	+	Dystonia, orobuccal movements	A, H, Fo, N, Fa	Moderate
16	64	M	−	Dystonia, athetosis	A, Le, H, N	Severe
17	61	M	+	None	A, Le, Fo	Severe
18	43	F	+	Orobuccal movements	A, Le, H, Fo, Fa	Moderate
19	65	F	−	Dystonia	A, Le, N	Severe
20	47	F	+	Oromandibular—lingual movements	A, H, Fo, Fa	Severe
21	76	F	+	None	A, Le, H	Severe
22	70	F	+	Orobuccal movements	A, H, Fa	Moderate
23	65	F	+	Facial grimacing	A, Le, Fa, H, Fo	Severe
24	31	M	−	Myoclonus, dystonia	A, Le	Severe
25	72	M	−	None	A, Le	Severe

A = arm; Le = leg; H = hand; Fo = foot; N = neck; Tr = trunk; Fa = face; F = female; M = male.

patients had bilateral ballistic movements from the beginning of the disease (patient 4 with encephalitis and patient 23 with systemic lupus erythematosus). In others, the right side was slightly more often affected (13 cases) than the left (10 cases). An arm and leg were simultaneously involved in 19 patients, whereas in five only the arm was affected. One patient (patient 1) had only ballistic movements of the leg (table 1).

Only two patients had "pure" hemiballism (table 1), without other types of involuntary movements. Ipsilateral hemichorea was associated with hemiballism in 16 patients, whereas facial and oromandibular-lingual dyskinesias, occasionally impairing speech and deglutition, were found in 11 patients. Nine patients had periodic, prolonged dystonic postures, usually of the head and trunk. Two patients developed parkinsonism. Patient 4, with encephalitis, developed postural instability, bradykinesia, and mild rigidity after ballistic movements subsided, but, except for persistence of slight postural instability, they disappeared in four months; this patient was treated with haloperidol, which might have provoked the parkinsonian signs. In patient 7, after a latency of two years, hemiparkinsonism was expressed on the opposite side to the initial hemiballism. Patient 21 was diagnosed initially as having left hemiparkinsonism with subsequent generalisation in the course of one year, three years before manifestation of the left sided hemiballism. During the follow up for hemiballism, parkinsonian symptoms were thereafter reported only on the right side of her body.

Involuntary movements caused severe disability in 17 patients (68%; table 1). Associated pyramidal weakness was registered ipsilaterally in nine patients and contralaterally in three. Hypotonia of the affected limbs was reported in four cases and hemihypaesthesia in three.

Ischaemic stroke was identified as a cause of abnormal movements in 10 patients (table 2), whereas in another six we considered stroke as a likely cause ("assumed stroke"). The average age at onset in this group was 62 (range 19–80) years. In patients 11 and 25 massive haemorrhage was found in diencephalic regions. In patient 18 hemiballism was the clinical manifestation of two transient ischaemic attacks, with durations of 45 and 100 minutes. Patients 17 experienced several such attacks before the abrupt onset of ballistic movements. Patients 20 had already had four episodes of hemiballism on the same side of the body during the five year follow up. Ballistic movements in this group began abruptly in 15 patients. In the remaining four they progressivelly worsened over a period of 10 days to 2·5 months. Diabetes mellitus was found in seven patients (five of them were insulin dependent), and 12 patients had arterial hypertension. Other risk factors included absolute arrhythmia in four and hyperlipidaemia in three patients.

Other causes identified in six patients (24%) were encephalitis, Sydenham's chorea, systemic lupus erythematosus, tuberous sclerosis, non-ketotic hyperglycaemia, and multiple punctiform calcifications of basal ganglia. The mean age at onset in this group was 37·5 (range 15–65) years (table 2). The young female patient with Sydenham's chorea (patient 15) had her third episode of ballistic movements—the first being during childhood and the second during pregnancy—eight years before admission to our Department. All patients in this group had an insidious onset of ballism (except the patient with systemic lupus erythematosus), with gradual progression over a period from two days to one month.

Studies with CT or MRI were performed in 22 patients. A contralateral lesion of the subthalamic nucleus was an isolated finding in one patient (patient 6). Simultaneous lesions of the subthalamic nucleus and other parts of the CNS (basal ganglia, pons, midbrain) were reported in five more patients (table 2). Different basal ganglia lesions were found in five patients and isolated thalamic haemor-

Table 2 Cause of hemiballism, response to treatment and CT and MRI findings in patients with ballistic movements

Patient	*Probable cause*	*Side of ballism*	*"Effective" drugs*	*Latency* (days)*	*CT or MRI findings*
1	Assumed stroke	R	HDL, CLON	33	Not performed
2	Assumed stroke	L	HDL	10	Not performed
3	Stroke	L	HDL, CLON	41	Multiple bilaterial ischaemia lesions in basal ganglia, pons, and right midbrain with affected right subthalamic nucleus; ventricular and sulcal enlargement
4	Encephalitis	Bilateral	HDL, COR	4	Normal
5	Assumed stroke	L	HDL	No response	Not performed
6	Stroke	L	HDL	6	Right subthalamic nucleus infarct
7	Stroke	R	HDL,TB	4	Multiple, bilateral lucency areas in the heads of caudate nuclei (L > R)
8	Stroke	R	HDL	8	Sulcal, as well as lateral and third ventricle enlargement, loss of substance in left diencephalon (lacunar infarcts?)
9	Stroke	R	HDL,TB	12	Two low density areas in the left frontal lobe and left basal basal ganglia
10	Non-ketotic hyperglycaemia	L	HDL	14	Haemorrhagic infarct in left(?) temporal lobe
11	Haemorrhage	R	HDL	2	Left thalamic, subthalamic nucleus, and pontine haemorrhagic lesions
12	Stroke	R	HDL, CLON	37	Multiple small lucency areas, also affecting both sides of diencephalon
13	Stroke	R	HDL, RS	No response (death after 16 days)	Normal
14	Basal ganglia calcification (vasculitis)	R	HDL	32	Multiple calcifications of the left basal ganglia
15	Sydenham's chorea	L	DZP	17	Bilateral enlargement of sulci; otherwise normal
16	Assumed stroke	R	HDL	19	Normal
17	Stroke	R	CHP	10	Massive infarct in left basal ganglia
18	Transient ischaemic attack	L	—	—	Normal
19	Assumed stroke	L	CHP	22	Normal, except for slight enlargement of sulci
20	Stroke	L	CHP	34	Right parietotemporal lucency
21	Stroke	L	HDL	6	Right basal ganglia infarct
22	Assumed stroke	R	HDL	8	Normal
23	CNS lupus	Bilateral	HDL, TB	Initial response after 3 days, but death after 14 days due to massive intracerebral haemorrhage	Large right parietal lucency
24	CNS tuberous sclerosis	R	CLON, HDL, PIM, DZP	No response	Multiple subependymal nodular calcifications and one in the region of the left subthalamic nucleus, with ventricular dilatation
25	Haemorrhage	R	CHP, DZP, RS	No response (death after 33 days)	Left thalamic haemorrhage

*Latency: considered as the period between the initiation of treatment and disappearance of ballistic movements. R = right; L = left; HDL = haloperidol; CLON = clonazepam; PIM = pimozid; DZP = diazepam; TB = tertrabenazine; RS = reserpine; CHP = chlorpromazine; COR = corticosteroids.

rhage in one patient. In three patients ischaemic lesions were noted in parietal regions, temporal lobe regions, or both, and in the remaining seven studies were normal (table 2).

Studies on CSF showed a mild to moderate increase in protein concentrations in six patients. No intrathecal synthesis of IgG, or the presence of oligoclonal bands were detected.

In the patient with Sydenham's chorea the antistreptinolysin antibody titre in serum was positive, with an increase in concentration in all three classes of immunoglobulins. Patients 23 with verified systemic lupus erythematosus met the criteria for the presence of lupus anticoagulant and also had increased serum immune complexes. The patient with encephalitis had raised IgE and IgM serum concentrations and decreased concentration of the complement C3 component. Two patients with stroke had hyper-γ-globulinaemia and increased immune complexes, with extremely high serum creatine phosphokinase activity in one of them.

We found complete disappearance of ballistic movements in nine patients. Five of them were treated with haloperidol only, the daily dose not exceeding 15 mg. Diazepam was used as monotherapy in the patient with Sydenham's chorea. In the remaining three patients, clonazepam, tetrabenazine, reserpine, and diazepam were used in combination with haloperidol. Complete remission was achieved after 6–37 (mean 15) days. In another seven patients the same therapeutic approach resulted in considerable relief of ballistic movements, with persistance of residual dyskinesias of various types, mainly chorea. Treatment was without effect in four patients (16%); two of them died, one from cardiac failure (patient 13) and one from repeated subarachnoid haemorrhage (patient 25). Patient 23, with systemic lupus erythematosus, responded favourably after three days to haloperidol and tetrabenazine, but two weeks later suddenly died after a massive intracerebral haemorrhage (table 2).

Maintenance treatment was continued for two to four months and then slowly tapered. In one patient ballistic movements reappeared six days after the treatment was interrupted. These were again fully controlled 13 days after reintroduction of haloperidol.

Discussion

Close association between hemiballism and lesions of the contralateral subthalamic nucleus was recognised by the turn of the century.[12 13] This association was supported by the experimental hemiballism in monkeys induced by injections of a γ-aminobutyric acid antagonist into the basal ganglia (pallidus/subthalamic nucleus).[14 15] Our finding that a lesion of the subthalamic nucleus was reported in six patients (24%), only one of whom had an isolated infarct of this region

(table 2), is in accordance with the clinical experience that lesions exclusively confined to the subthalamic nucleus in humans are relatively rare.[16] Moreover, the neuroimaging studies were normal in seven cases (28%), although the possibility that small lacunar infarcts in the subthalamic nucleus or other parts of the basal ganglia might have escaped detection could not be ruled out.[8] In two patients MRI indicated multiple lacunar infarcts dispersed throughout the brain, involving also the region of the basal ganglia and subthalamic nucleus. A pathophysiological explanation for hemiballism due to the subthalamic nucleus lesion proposed the loss of an excitatory subthalamic influence on the internal segment of the globus pallidus.[1 17]

Cases with hemiballism were more often associated with a lesion that affected the afferent and efferent pathways of the subthalamic nucleus, or its projection areas,[18–20] as well as the striatum,[9 20–22] thalamus,[6 23] and the cerebral cortex.[24] Our finding of contralateral or bilateral lesions of basal ganglia in five, and a thalamic lesion in one patient are in concert with these reports. It is, however, impossible to exclude the possibility that the subthalamic nucleus was involved as well as the mentioned areas, on the basis of neuroimaging studies only. Hyland and Forman[23] postulated that lesions sparing the subthalamic nucleus but possibly involving its afferent pathways might give a better chance for spontaneous remission of ballistic movements, and Lang[9] suggested that lesions outside the subthalamic nucleus, possibly affecting the striatum, could negatively interfere with the adaptation mechanisms important for the spontaneous resolution of the ballistic movements. In our patients with persistent hemiballism no striatal pathology was seen on CT (table 2).

Most lesions that induce hemiballism are of vascular origin.[2 3 6] Ischaemic and haemorrhagic strokes were identified as a cause of involuntary movements in 18 patients (72%), which is in accordance with the findings in previous reports.[2 3 23] The suggestion that virtually any structural lesion, if properly situated, could induce hemiballism was confirmed by its association with metastatic tumours,[25–27] multiple sclerosis,[28 29] head trauma,[23 30] tuberculoma,[31] systemic lupus erythematosus,[32] scleroderma,[4] transient ischaemic attacks,[31 33] subarachnoid haemorrhage,[4] thalamotomy,[34 35] syphilis,[6] acquired immune deficiency syndrome,[5] hyperglycaemia,[36] arteriovenous malformations,[10 37] cerebral toxoplasmosis,[38] neurodegenerative diseases,[39] cystic glioma of the midbrain,[5] basal ganglia calcifications,[40] as well as during the administration of oral contraceptives,[41] levodopa,[42] phenytoin,[43] and neuroleptics.[44] In 21 patients with hemiballism-hemichorea, Dewey and Jankovic[5] reported an aetiology other than stroke in 10 patients. Our results also indicate a considerable percentage of patients with different aetiological factors causing hemiballism (28%).

Aetiological factors we identified in our patients have already been described,[15] except the central tuberous sclerosis (patient 24; table 2). Although the basal ganglia region is often affected in tuberous sclerosis, clinical evidence of dyskinesias is rare, except for two reported patients with chorea.[45 46]

The mean age at onset of hemiballism varied depending on the underlying aetiology, being between 64 and 75 years in the studies where in all or most cases the cause of hemiballism was stroke.[2 3 6] In the group of Dewey and Jankovic,[5] where about half of the patients had aetiological factors other than stroke, the mean age at onset was much lower (48 years). Our data give a mean value between these two (58·9 years), although we also found considerable difference between the patients with hemiballism due to stroke (66·9 years) and those with other aetiological factors (33·8 years; tables 1 and 2). In two cases bilateral ballism (biballism) was found. Biballism is extremely rare; Shannon[1] reviewed fewer than 20 cases in the literature, due to multiple sclerosis, basal ganglia calcification, systemic lupus erythematosus (as in patient 23), bilateral haemorrhage in the basal ganglia, as familial bilateral ballism, and phenytoin intoxication, whereas we report our case with encephalitis (patient 4).

We found only two cases of "pure" hemiballism, without the presence of other kinds of abnormal movements, whereas 16 patients (64%) had associated ballistic and choreic movements, which is in accordance with the recent data of Dewey and Jankovic.[5] Moreover, after spontaneous or drug induced cessation of ballistic movements, chorea persisted in seven patients, suggesting that these two types of involuntary movements represent "a spectrum of the same basic disease processes".[5]

An especially intriguing finding was the clinical relation of parkinsonism and hemiballism in patient 21. The diagnosis of Parkinson's disease was established three years before the occurrence of hemiballism on the same side as the initial parkinsonian signs. After the cessation of ballistic movements parkinsonian signs were repeatedly reported only on the contralateral side, whereas they disappeared on the side affected by hemiballism. Sellal *et al*[47] recently described a man with Parkinson's disease who suddenly developed left hemiballism due to a haematoma of the right subthalamic nucleus. After the ballistic movements had disappeared, akinesia and the other parkinsonian signs did not reappear on the left, providing clinical evidence for the suggested pivotal role of overactivity of the subthalamic nucleus in Parkinson's disease.[48–50] The CT findings in our patient, however, showed a right basal ganglia infarct without selective involvement of the subthalamic nucleus, implicating different mechanisms in the control of parkinsonian signs.

The prognosis of our patients was good, with complete recovery in nine; in seven more patients the abolishment of ballistic movements was accompanied by persistent but functionally irrelevant chorea. Three patients died. Hence, our experience favoured the

statement that good prognosis in hemiballism is an expected outcome rather than the exception.[1] We were unable to distinguish spontaneous improvement from the improvement induced by medication, as in all the patients (except patient 18 with transient ischaemic attacks) treatment started on admission to the department. Treatment was based on neuroleptics, mainly haloperidol, and in a few patients chlorpromazine and pimozid. We were unable to estimate the reported efficacy of tetrabenazine,[51] reserpine,[52] and clonazepam,[5] as these drugs were given in parallel with neuroleptics. In one patient, who experienced reappearance of ballistic movements with the discontinuation of haloperidol, and full control after reinstitution of the treatment, we could evidence the drug induced clinical improvement. The beneficial response to dopamine receptor blocking agents has been claimed to be usually dramatic and almost always within seven days,[1] although we found a longer delay with similar regimens (mean 15 days).

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